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REGRESSION OF CORONARY ATHEROSCLEROSIS: A PROSPECTIVE, QUANTITATIVE ANGIOGRAPHIC STUDY

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To assess the incidence and pattern of spontaneous coronary regression, we measured 942 stenoses $\geq 40\%$ from 305 pts who had 2 arteriograms 2 years apart in a trial where the intervention had no effect on established lesions. A radiologist, blinded to the order of the films, viewed them together to select frames for quantitative measurement. The CAAS system of Reiber et al was used. Regression $\geq 20\%$ was classified as definite and $\geq 10 < 20\%$ as borderline:

Severity	Number of lesions	REGRESSION		
		Borderline	Definite	Total
40-49%	344	25 (7.3%)	6 (1.7%)	31 (9.0%)
50-59%	225	13 (5.8%)	5 (2.2%)	18 (8.0%)
60-69%	103	16 (16%)	4 (3.9%)	20 (19%)
70-89%	59	3 (5.1%)	5 (8.5%)	8 (14%)
90-99%	71	1 (1.4%)	12 (17%)	13 (18%)
100%	140	3 (2.1%)	21 (15%)	24 (17%)
Total	942	61 (6.5%)	53 (5.6%)	114 (12.1%)

Morphological features did not predict regression, but the prevalence of frank intracoronary thrombus and irregular lesions were both less than 3%. Regression was borderline in 54 (78%) of the 69 stenoses $< 70\%$ but was definite in 38 (84%) of the 45 stenoses $\geq 70\%$ ($p < 0.0001$).

A potential explanation for these findings is that regression of severe stenoses results from thrombus remodeling and that regression of mild lesions represents either "true" regression or measurement error. Overall, 12% of lesions regress in 2 years.

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THE NATURAL HISTORY OF CORONARY ATHEROSCLEROSIS USING QUANTITATIVE ANGIOGRAPHY: IMPLICATIONS FOR REGRESSION TRIALS

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The Harvard Atherosclerosis Reversibility Project (HARP) pilot study was undertaken to study the natural history of coronary atherosclerosis progression using quantitative angiography. 26 patients with 76 diseased arterial segments underwent cardiac catheterization a mean of 3.0 years apart. Cholesterol was lowered more in the niacin group (-35.9 mg%, $n=9$) compared with the AHA diet ($+5.9$ mg%, $n=8$, $p=.0001$) or the intensive dietary group (-4.6 mg%, $n=9$, $p=.002$). There were no significant differences among the 3 treatment groups in the primary endpoint, the change in minimum diameter (MD) analyzed as a continuous variable on either a per patient basis or a per segment basis. Paradoxically, percent stenosis (%S) progressed more ($p=.009$) in the niacin group ($+5.5\%$) than in the intensive dietary group (IDG) which regressed by -0.7% . This apparent "regression" in %S in the IDG was artifactual and due to a slightly more rapid rate of progression in the "normal" reference segment ($+13$ mm) compared with the minimum diameter ($+3$ mm). Overall, mild lesions progressed more rapidly than severe lesions ($p=.00009$ for MD, $p=.009$ for %S). Stenoses within a patient progressed at rates that were independent of each other ($r=-.09$ for MD, $r=-.005$ for %S) after adjusting for initial severity and treatment group assignment. The MD of stenoses proximal to bypass graft insertion progressed more rapidly ($p=.057$) than those distal to graft insertion. Conclusions: 1. Mildly narrowed coronary arteries progress more rapidly than severe lesions, therefore initial severity must be taken into account. 2. Occasionally the adjacent "normal" reference segment progresses more rapidly than the minimum diameter causing %S to be a misleading measure of disease progression. 3. Within a given patient, segments change in diameter nearly independently of each other when analyzed as a continuous variable. 4. Local coronary hemodynamics may influence the rate of atherosclerosis progression.

11:45

CORONARY DISEASE: IS THE SPEED OF SUBSEQUENT PROGRESSION PREDICTABLE FROM ANGIOGRAPHY? RESULTS OF INTACT

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To test the hypothesis, that the coronary angiogram (CA) indicates future speed of progression in patients (pts) with coronary disease, we compared results of 2 standardized CA's, repeated after an interval of 3 years in 348 INTACT pts. Both CA's were quantitatively analyzed with an automatic edge detection system (CAAS). At 1. CA 29 pts had no coronary lesions (L; = stenoses $\geq 20\%$ or occlusions) apart from previously dilated stenoses (group (Gr) A), 134 pts had 1-2 L (Gr B), 79 pts 3-4 L (Gr C), 58 pts 5-6 L (Gr D), 28 pts 7-9 L (Gr E) and 20 pts > 9 L (Gr F). For these groups the no. of classical risk factors (RF; = smoking, hypercholesterinemia, hypertension), the no. of progressing stenoses ($\geq 20\%$; PRO) and the no. of newly formed lesions (NL) seen at 2. CA are listed (mean values/pat); Cochran's linear trend test: $p < 0.001$:

Gr	A	B	C	D	E	F
RF	1.56	1.59	1.55	1.56	1.79	1.33
PRO	0.00	0.11	0.42	0.57	0.86	0.75*
NL	0.41	0.59	0.73	0.81	0.93	1.25*

Conclusion: 1) With increasing no. of lesions at the 1. CA pts are prone to a higher speed of future progression. Pts with a high risk of progression should be followed up closely and liberally be considered for reangiography. 2) The no. of classical risk factors is not different between pts with a high or low progression rate during 3 years.

Wednesday, March 6, 1991

10:30AM-12:00NOON, Room 260, West Concourse
Catheter Ablation of Accessory Pathways II

10:30

CATHETER PLACEMENT FOR RADIOFREQUENCY ABLATION OF ANTEROSEPTAL ACCESSORY PATHWAYS. Nicholas Twidale, Xunzhang Wang, Kriegh Moulton, Karen Beckman, Michael Prior, Andrew Hazlitt, Ralph Lazzara, Warren Jackman. Univ. of Oklahoma and VAMC, Oklahoma City, OK.

Catheter ablation of anterosseptal (AS) accessory pathways (AP) may be complicated by AV block. Believing the His bundle (H), surrounded by fibrous tissue, to be more resistant to injury than the AV node, radiofrequency energy (RF) was applied to the ventricular (V) side of the tricuspid (T) annulus in 8 patients (pts) with AS APs. APs were defined as AS if both AP and H potentials were recorded from the same 2 mm-spaced bipolar electrode. AP conducted only retrogradely in 2 pts. A 7F catheter with a 4 mm large tip electrode (LTE) was inserted via a subclavian vein, advanced into the right V, curved 180° and withdrawn beneath the T leaflet until the LTE was held firmly against the T annulus and recorded AP potential. RF (32 ± 6 Watts for 41 ± 18 sec) applied to the LTE eliminated AP conduction in 6/8 pts. In the remaining 2 pts, AP conduction was eliminated by RF (25 ± 1 Watts for 33 ± 38 sec) delivered through a LTE positioned parallel to the H catheter and recording AP potential. Of 6 pts with antegrade AP conduction, preexcitation and reentrant tachycardia (RT) have not returned in 426 mths of followup. In the 2 pts with concealed APs, AP conduction and RT recurred but AP was successfully re-ablated 2 wks and 7mths following the original procedure. Although right bundle branch block was produced in 5 pts, block distal to H did not occur during atrial pacing and 1:1 AV nodal conduction was preserved (352 ± 64 ms pre, 310 ± 59 ms post-ablation). We conclude: 1) APs in close proximity to H can be ablated by RF with low risk of AV block; 2) placement beneath T leaflet ensures V location and firm contact.